Treatment of Subarachnoid Hemorrhage

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KEYWORDS

• Subarachnoid hemorrhage • Vasospasm • Delayed cerebral ischemia • Aneurysm
• Transcranial Doppler ultrasonography

KEY POINTS

- Subarachnoid hemorrhage is typically caused by a ruptured intracranial aneurysm and presents with a sudden severe, headache often accompanied by syncope, nausea, and vomiting.
- Initial management includes airway assessment, blood pressure control, treatment of pain, and noncontrast computed tomography (CT), followed by urgent catheter or CT angiography.
- To reduce the risk of delayed cerebral ischemia (DCI), all patients should be treated with nimodipine and be maintained in a euvolemic state in the days after hemorrhage.
- The development of narrowing of large cerebral vessels (vasospasm) can be detected with transcranial Doppler ultrasonography, CT, or conventional angiography. Vasospasm is closely correlated with DCI, but each can occur independently.
- DCI can be treated with combinations of blood pressure or cardiac output augmentation, angioplasty of proximal vasospastic vessels, and selective intra-arterial infusions of vasodilators.

INTRODUCTION

Nontraumatic subarachnoid hemorrhage (SAH) typically presents as a sudden severe headache, often described as "the worst headache of my life." Consciousness may be impaired, but focal neurologic deficits are uncommon. The incidence of SAH ranges from 10 to 18 per 100,000 people.[1] It often occurs in middle-aged patients and has a female predominance.[2] Of all spontaneous SAHs, 80% are the result of the rupture of an intracranial aneurysm, 15% do not have a bleeding source identified, and the remainder are owing to a myriad of other causes, mostly vascular malformations, but also vasculitis or posterior reversible vasoconstriction syndrome. Genetic factors seem to play a role in some families.

Disclosures: None.
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Crit Care Clin 30 (2014) 719–733
http://dx.doi.org/10.1016/j.ccc.2014.06.004 **criticalcare.theclinics.com**
0749-0704/14/$ – see front matter

Intracranial berry (ie, saccular) aneurysms are typically found near the circle of Willis at the branching points of large cerebral arteries. Hemodynamic stress at arterial branching sites and inflammation seem to contribute to aneurysm formation. About 2% to 5% of the population harbor intracranial aneurysms.[3] Although aneurysms are thought to develop over many years, cases of rapid growth in size do occur. They frequently arise off the internal carotid artery at the take-off of the anterior and posterior communicating arteries and middle cerebral artery. Relatively few occur in the posterior circulation. Risk factors for aneurysmal rupture include smoking, hypertension, alcohol use, and having first-degree relatives with SAH.[4,5] Autosomal-dominant polycystic kidney disease is the most common heritable disorder to increase the risk for SAH; others include connective tissue disorders.

PATIENT EVALUATION

The classical clinical triad for presentation of SAH includes sudden severe headache, syncope, and vomiting. Other common symptoms include nausea, photophobia, and altered consciousness. Focal neurologic deficits occur in about 10% of patients. They can be owing to aneurysmal compression of a cranial nerve (typically a posterior communicating artery aneurysm compressing the third nerve producing ptosis, a dilated pupil, and limited medial and vertical gaze). More ominous are focal deficits owing to thick subarachnoid clots or parenchymal hematoma. Blood released under high pressure may directly cause damage to local tissues. Additionally, the vessel rupture produces an increase in intracranial pressure (ICP) that approaches arterial pressure and cerebral perfusion falls to nil. If hemorrhage stops and the acute rise in ICP is transient, it can result in nausea, vomiting, and syncope; if the high pressure is sustained, it is uniformly fatal. Exposure of the meninges to blood causes irritation resulting in photophobia, neck stiffness, and eventually back pain. Blood pressure is frequently elevated, which may increase the risk of re-rupture.

Initial Stabilization

Initial management should focus on airway management in comatose patients and blood pressure control to stabilize the patient, with the goal of obtaining a computed tomography (CT) scan as soon as possible (**Box 1**).

Patients may be unable to protect their airway and require intubation for multiple reasons, including hydrocephalus, seizure, or sedation. In addition, elective intubation may be necessary in agitated patients to safely and expeditiously perform cerebral angiography.

To reduce the risk of rebleeding before the aneurysm is secured, blood pressure should be maintained at the patients baseline levels or, if unknown, a mean arterial blood pressure of less than about 110 mm Hg. Effective pain control may be sufficient to manage blood pressure; otherwise short-acting intravenous medications (eg,

Box 1
Initial stabilization and evaluation

- Airway assessment in comatose patients
- Control hypertension
- Treat headache
- Noncontrast CT scan as soon as possible
- Lumbar puncture if CT negative and suspicion high